Original Article

Fasciocutaneous sural nerve flap for lower extremity reconstruction: include or exclude the sural nerve?

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Abstract: Our purpose was to compare outcomes of distally-based sural fasciocutaneous island flaps with and without inclusion of the sural nerve. The records of patients with defects in the foot and ankle that received distally-based sural fasciocutaneous island flaps with or without preservation of the sural nerve from June 2006 to July 2013 were reviewed. Classification of sensory recovery was based on the British Medical Research Council scale (BMRC, 1954). Twenty-three patients (23 flaps) were included, the majority was male (73.9%), and the mean age was 46.1 ± 14 years. Wound size ranged from 3×3 cm to 20×14 cm. Flap size ranged from 6×4 cm to 22×15 cm, and the median length of the flap pedicle was 10 ± 3.3 cm. Of the 23 patients, 14 had sural nerve preservation. Flap pedicle length was similar in the 2 groups, but the pivot point was more distal (5.7 cm vs. 4.0 cm, \( P = 0.020 \)) and the flap area smaller (80.7 cm\(^2\) vs. 159.8 cm\(^2\), \( P = 0.024 \)) in the nerve preservation group. Flap survival was similar between the groups. Recovery of skin sensation was better in the sural nerve preservation group (\( P < 0.001 \)); about 89% of patients in the traditional surgery group had numbness whereas approximately 50% of patients in the sural nerve preservation group regained normal skin sensation. In conclusion, preservation of the sural nerve is feasible during the creation of a distally-based sural artery flap, and can eliminate loss of sensation of the lateral foot.

Keywords: Ankle, flap, foot, perforator, sural nerve

Introduction

Coverage of the soft tissue defects in the foot and ankle remains a difficult problem for surgeons, largely due to the scarcity of muscle and skin. A variety of options are available, and all have certain benefits and limitations [1-7]. Free flaps enable transplantation of a large amount of tissue with single-stage reconstruction, but requires microsurgical experience and a long surgical time. Distally-based sural nerve island flaps were reported in the early 1990’s by Masquelet et al [8], and were subsequently popularized and refined [9].

A distally-based sural fasciocutaneous flap has the advantages of a reliable blood supply, easy and fast elevation, preservation of the major arteries of leg, and allows one-stage reconstruction. This flap does not require microsurgical expertise and equipment, does not sacrifice a major artery, and minimizes donor site injury [5]. However, the sural nerve is generally sacrificed in creation of the flap resulting in sensory loss to the lateral side of the foot. This can result in decreased patient satisfaction, even if they have been made aware of the potential loss of sensation preoperatively.

Since 2003 we have been preserving the sural nerve [10-13] in select cases of distally-based sural nerve island flaps created for foot and ankle reconstruction. The purpose of this study was to present the results of flaps in which the sural nerve was preserved, compare the results to those of when the sural nerve was ligated, and discuss the criteria for cases in which preservation of the sural nerve is appropriate.

Patients and methods

The records of patients with skin and tissue defects in the foot and ankle that were reconstructed with distally-based sural fasciocutane-
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ous island flaps with or without preservation of the sural nerve from June 2006 to July 2013 were retrospectively reviewed. Sural nerve preservation was not considered in patients at high risk for flap complications, in whom a relatively long flap pedicle was required, or in whom dissection of the sural nerve was judged to be difficult upon intraoperative examination.

Surgical technique

Because there is large individual variation in the course of the sural nerve, it is impossible to identify variations of the nerve and blood vessels before surgery. After administration of epidural anesthesia, the patient was placed in the prone position. Using Doppler ultrasonography, the lowermost septocutaneous perforator of the peroneal artery was detected, and marked at 5 cm above the lateral malleolus in the lateral retromalleolar region. A line was drawn from the middle popliteal fossa to the midpoint between the Achilles tendon and lateral malleolus, which is the course of the sural nerve and the lesser saphenous vein. The skin was marked along the midline of the posterior aspect of the leg based on the size of the soft-tissue defect. With the aid of a tourniquet, the subdermal layer was dissected to expose the lesser saphenous vein, sural nerve, and the accompanying sural vessels in pedicle area. The flap was then raised from the subfascial plane in a proximal-to-distal direction. When dissecting the proximal flap, both the lateral and medial branches of sural nerve were carefully protected. Using a magnifying glass, the sural nerve was carefully dissected along the sural vessels and excluded from the flap until the pivot point. The lesser saphenous vein was dissected if an anastomosis was to be performed, but if the vein was injured or no anastomosis was needed, the proximal end was ligated and cut. The flap was rotated 180° to reach the recipient site, and inserted without tension. Temporary anchoring stitches were used to secure the deep fascia.

Over the flap fascial pedicle, a large Z-plasty skin extension was created as the roof of the cutaneous tunnel to decrease tension on the pedicle. If the sural flap was large and the pedicle was long, a fasciocutaneous pedicel not less than 3 to 4 millimeters in width was used. A semi-circular incision was made at the pivot point.

Postoperative care and follow-up

After surgery, the affected limb was kept raised in order to prevent compression of the pedicle. Dextran-40, anisodamine, and corticosteroids were administered by intravenous infusion for 3 to 5 days. If venous congestion was noted, a mini-incision to reduce pressure was made, and a wet heparin compress was applied. Sensation was detected in the lateral side of the foot in all patients upon waking from anesthesia.

All patients were followed for a minimum of 3 months after surgery. During follow-up visits, pin prick testing was performed to determine sensory function. Classification of sensory recovery was based on the British Medical Research Council scale as follows: S0: No recovery of sensation in the area of the nerve; S1: Recovery of deep cutaneous pain sensibility in the area of the nerve; S2: Recovery of superficial pain and some touch sensibility; S2+: As in S2, but with an exaggerated response; S3: Recovery of pain and touch sensibility with the absence of exaggerated response, and 2-point discrimination (2PD)>15 mm; S3+: As in S3, but good localization of the stimulus and imperfect recovery of 2PD (7-12 mm); S4: Complete recovery, 2PD<7 mm.

Statistical analysis

Continuous data including patient age, pivot point, length of flap pedicle, and flap area were reported as mean and standard deviation. Other categorical variables were reported as count and percentage. Independent t-test was applied for examining the difference in continuous variables between patients who received surgery with and without sural nerve preservation, and chi-square test or Fisher’s exact test was applied for categorical variables. The change of sensory recovery during 6 months of follow-up was summarized as number and percentage. A value of $P<0.05$ was considered to indicate statistical significance. All statistical analyses were 2-sided, and performed using SPSS 22.0 statistics software (IBM Corp., Armonk, NY, USA).

Results

A total of 23 patients who received 23 flaps were included in the analysis (Table 1). The
# Table 1. Patient data

<table>
<thead>
<tr>
<th>No</th>
<th>Age/ Sex</th>
<th>Flap outcome</th>
<th>Cause of defect</th>
<th>Location</th>
<th>Pivot point (cm)</th>
<th>Pedicle length (cm)</th>
<th>Pedicle width (cm)</th>
<th>Defect Size (cm)</th>
<th>Flap size (cm)</th>
<th>Sural nerve preservation</th>
<th>Postoperative skin sensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Distal anterior leg</td>
<td>6</td>
<td>9.5</td>
<td>2</td>
<td>5×4</td>
<td>6.5×6</td>
<td>Interior (medial) and lateral branch, open tunnel</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>42/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Middle anterior leg</td>
<td>20</td>
<td>9</td>
<td>3</td>
<td>6×4</td>
<td>7×5</td>
<td>Sparing main branch</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>40/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Dorsal and forefoot</td>
<td>4</td>
<td>14</td>
<td>2; 4</td>
<td>14×9</td>
<td>16×11</td>
<td>No</td>
<td>Numbness of lateral ankle area after surgery, but the area of numbness decreased by 1/3 at 6-month follow-up. Lateral area hypersensitive postoperatively; normal after 3 months. Decreased sensation in lateral malleous area postoperatively, but resolved after 1 month.</td>
</tr>
<tr>
<td>4</td>
<td>33/M</td>
<td>Tip necrosis</td>
<td>Crush</td>
<td>Forefoot</td>
<td>6</td>
<td>18</td>
<td>2.5</td>
<td>11×7.5</td>
<td>13×9</td>
<td>Lateral branch</td>
<td>Normal</td>
</tr>
<tr>
<td>5</td>
<td>58/M</td>
<td>Tip necrosis</td>
<td>Traffic accident</td>
<td>Forefoot</td>
<td>5</td>
<td>2; 3</td>
<td>14×9</td>
<td>16×11</td>
<td>Lateral branch</td>
<td>Decreased sensation in lateral malleous area postoperatively; normal after 6 months. Normal after 2 weeks. Lost to follow-up.</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>40/F</td>
<td>Tip necrosis</td>
<td>Traffic accident</td>
<td>Dorsal foot</td>
<td>5</td>
<td>14</td>
<td>4</td>
<td>7×3</td>
<td>8×4</td>
<td>No</td>
<td>Sensory loss at lateral foot postoperatively. Lost to follow-up.</td>
</tr>
<tr>
<td>7</td>
<td>82/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Heel</td>
<td>4</td>
<td>7</td>
<td>1.5</td>
<td>3×3</td>
<td>5×5</td>
<td>Interior branch</td>
<td>Normal</td>
</tr>
<tr>
<td>8</td>
<td>58/M</td>
<td>Tip necrosis</td>
<td>Traffic accident</td>
<td>Planta</td>
<td>5</td>
<td>9</td>
<td>1.5</td>
<td>18×14</td>
<td>Sparring</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>26/F</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Medial heel</td>
<td>4</td>
<td>9</td>
<td>3</td>
<td>6×4</td>
<td>7.5×5.5</td>
<td>Sparing</td>
<td>Normal</td>
</tr>
<tr>
<td>10</td>
<td>56/F</td>
<td>Complete survival</td>
<td>Crush, electric burn</td>
<td>Medial ankle</td>
<td>7</td>
<td>9</td>
<td>2; 4</td>
<td>6×6</td>
<td>8×8</td>
<td>Interior branch (passive)</td>
<td>Normal</td>
</tr>
<tr>
<td>11</td>
<td>50/M</td>
<td>Complete survival</td>
<td>Chronic osteomyelitis</td>
<td>Heel</td>
<td>5</td>
<td>8</td>
<td>1.5×4</td>
<td>9×2</td>
<td>11.5×5</td>
<td>Sparring lateral branch</td>
<td>Normal</td>
</tr>
<tr>
<td>12</td>
<td>58/F</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Medial ankle</td>
<td>4</td>
<td>10</td>
<td>1.5×3</td>
<td>3.5×3.5</td>
<td>6×4</td>
<td>Interior and lateral branch</td>
<td>Normal</td>
</tr>
<tr>
<td>13</td>
<td>56/M</td>
<td>Complete survival</td>
<td>Crush</td>
<td>Dorsal and forefoot</td>
<td>3.5</td>
<td>12</td>
<td>3</td>
<td>11×7</td>
<td>13×9</td>
<td>No, dominant area loss</td>
<td>Sensory loss at lateral area (S0). Sensory loss zone reduced by 1/5 after 3 months. Decreased sensation in lateral malleous area. Area of decreased sensation reduced after 3 months. Sensory loss at lateral area (SO). Area of decreased sensation reduced after 6 months.</td>
</tr>
<tr>
<td>14</td>
<td>35/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Dorsal foot</td>
<td>3.5</td>
<td>6</td>
<td>3</td>
<td>18×12</td>
<td>20×13</td>
<td>No, dominant area loss</td>
<td>Sensory loss at lateral area (S0). Area of decreased sensation reduced after 6 months. Unchanged after 1 month. Sensory loss at lateral area (SO). Unchanged after 1 month.</td>
</tr>
<tr>
<td>15</td>
<td>39/F</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Dorsal foot</td>
<td>3.5</td>
<td>10</td>
<td>3</td>
<td>20×14</td>
<td>22×15</td>
<td>No, dominant area loss</td>
<td>Sensory loss at lateral area (S0). Area of decreased sensation reduced after 6 months.</td>
</tr>
<tr>
<td>16</td>
<td>36/F</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Medial ankle and foot</td>
<td>5</td>
<td>8</td>
<td>3</td>
<td>15×7</td>
<td>16×9</td>
<td>No</td>
<td>Sensory loss at lateral area (S0). Sensory loss zone slightly reduced after 3 months. Sensory loss at lateral area (S0). Sensory loss zone slightly reduced after 3 months. Sensory loss at lateral area (S0).</td>
</tr>
<tr>
<td>17</td>
<td>47/M</td>
<td>Tip necrosis</td>
<td>Traffic accident</td>
<td>Heel</td>
<td>4</td>
<td>6</td>
<td>3</td>
<td>13×7</td>
<td>15×9</td>
<td>Interior and lateral branch</td>
<td>Sensory loss at lateral area (SO). Normal after 5 months. Sensory loss at lateral area (S0). Normal after 7 months.</td>
</tr>
<tr>
<td>18</td>
<td>55/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Dorsal foot</td>
<td>4</td>
<td>6</td>
<td>3</td>
<td>20×8</td>
<td>18×9</td>
<td>No</td>
<td>Sensory loss at lateral area (S0). Unchanged after 1 month. Sensory loss at lateral area (S0). Unchanged after 1 month.</td>
</tr>
<tr>
<td>19</td>
<td>20/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Dorsal foot, medial ankle</td>
<td>3.5</td>
<td>14</td>
<td>2; 4</td>
<td>13×6</td>
<td>14×7.5</td>
<td>No</td>
<td>Sensory loss at lateral area (S0). Unchanged after 1 month. Sensory loss at lateral area (SO). Unchanged after 1 month.</td>
</tr>
<tr>
<td>20</td>
<td>34/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Dorsal foot</td>
<td>4</td>
<td>13</td>
<td>2; 3</td>
<td>14×6</td>
<td>15×7.5</td>
<td>No</td>
<td>Sensory loss at lateral area (S0). Sensory loss after 6 months. Unchanged after 1 month.</td>
</tr>
<tr>
<td>21</td>
<td>57/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Distal medial leg</td>
<td>5</td>
<td>13</td>
<td>2; 4</td>
<td>9×7</td>
<td>10×8</td>
<td>Interior and lateral branch</td>
<td>Decreased sensation in lateral malleous area. Area normal at 1 month after surgery. Decreased sensation at lateral foot. Area numb subjectively, but sensitive to needle test. Lateral foot hypersensitivity. Normal sensation at 1 month.</td>
</tr>
<tr>
<td>22</td>
<td>35/M</td>
<td>Complete survival</td>
<td>Traffic accident</td>
<td>Distal posterior leg</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>63/M</td>
<td>Complete survival</td>
<td>Local infection</td>
<td>Achilles tendon exposure</td>
<td>9</td>
<td>6</td>
<td>2; 3</td>
<td>5×3</td>
<td>6×4</td>
<td>Sparing</td>
<td>Normal</td>
</tr>
</tbody>
</table>

The first number is the width of the fascia pedicle, and the second number in the width of the superficial skin of the fascia pedicle. A single number indicates the width of the superficial skin of the pedicle is not available, and indicated with width of the fascia pedicle.
The majority of patients were males (73.9%), and the mean age was 46.1 ± 14 years (range, 20–80 years). Defects were the result of a traffic accident in 16 patients, crush injury in 3, infected ulcer or osteomyelitis in 3, and skin dry necrosis in 1. The defects were located in the proximal and mid-distal dorsal foot in 11 cases, heel in 4, medial malleolus in 4, Achilles tendon in 1, and distal leg in 3. All wounds were complicated by bone, tendon, and joint or internal fixation device exposure, and the size of the wounds ranged from 3×3 cm to 20×14 cm.

Flap size ranged from 6×4 cm to 22×15 cm, the median length of the flap pedicle was 10 ± 3.3 cm (range, 6–18 cm), and the width ranged from 2 to 4 cm. The distal pivot point was located at 3.5 cm above the top of the lateral malleolus. All donor site defects were resurfaced with split-thickness skin grafts and healed primarily.

Eighteen flaps survived completely (survival rate 78.3%) and the other 5 (21.7%) flaps had superficial necrosis. Four (17.4%) patients received split-thickness skin grafts, and all experienced partial flap necrosis.

Postoperatively, sural nerve skin sensation was S0 in 8 patients, S2+ in 3 patients, and S3+ to S4 in 12 patients (Table 2). Of patients with abnormal skin sensation, satisfactory sensory recovery (from S2+ or S3+ to normal) occurred in 5 patients, no change with nidus shrinkage was present in 6 patients, and numbness was present in 1 patient at 6 months after surgery (Table 3).

Of the 23 patients, 14 had sural nerve preservation and a comparison of patients with and
Fasciocutaneous sural nerve flap

without preservation is shown in Table 2. The 2 groups were similar in age and sex distribution. Defects on the dorsal aspect of the feet were more common in the traditional surgery group (88.9% vs. 0%), while defects in the sural nerve preservation group were more common in the distal feet, Achilles tendon, forefoot, heel, and planta (71.4% vs. 0%). The length of the flap pedicle was similar in the 2 groups, but the position of the pivot point was more distal (5.7 cm vs. 4.0 cm, \(P=0.020\)) and the flap area was smaller (80.7 cm\(^2\) vs. 159.8 cm\(^2\), \(P=0.024\)) in the sural nerve preservation group.

The proportion of flaps that survived was similar between the groups. Postoperative recovery of skin sensation was much better in the sural nerve preservation group (\(P<0.001\)); about 89% of patients in the traditional surgery group had numbness whereas approximately 50% of patients in the sural nerve preservation group regained normal skin sensation.

Discussion

The results of this study indicate that preservation of the sural nerve during the creation of a distally-based sural artery flap is feasible, and can reduce sensory loss and preserve sensation on the lateral side of the foot.

Coverage of the soft tissue defects in foot and ankle is problematic due to the scarcity of muscle and skin. While free flaps enable transplantation of a large amount of tissue in a single-stage reconstruction, microsurgical experience is required and the risk of flap loss is high. The distally-based sural fasciocutaneous flap has the advantages of single-stage reconstruction, a reliable blood supply, easy and fast elevation, and preservation of the major arteries of the leg. However, traditional use of the flap sacrifices the sural nerve and thus leads to loss of sensation of the lateral foot.

The sural nerve is made up of the medial branch of the tibial nerve and the lateral branch of the common fibular nerve. There is large variation of these 2 branches in thickness and are of skin innervation in the lateral side of the foot [14, 15]. In the early reports [1, 2, 8], the sural nerve was described as an essential structure for blood supply. But our anatomic study showed a relative safe distance between the axial arterial network and the sural nerve in the posterior calf [16]. We also demonstrated that the extrinsic vessels around the sural nerve and lesser saphenous vein (not including the intrinsic vessels of the sural nerve) can support flap survival in our series of distally-based sural flaps.

When the sural nerve is sacrificed, most patients complain of hyperesthesia or numbness of the lateral aspect of the foot. However, controversy still exists about whether the flap should include both the sural nerve and lesser saphenous vein along with the accompanying vessels, or if the sural nerve should be excluded. Some authors recommend raising the flap without the sural nerve [10, 11]. Masquelet et al [8], who first introduced the concept of neurocutaneous flaps, suggests taking the nerve with the pedicle. Hasegaw et al [3] also support nerve inclusion. Jeng et al [17, 18] advised leaving the sural nerve behind, and raising the lateral branch of the sural nerve with the flap and performing neurorrhaphy with the common sural nerve. However, the potential for injury to the common peroneal nerve exists. One study reported that sensation of the lateral foot recovered postoperatively [6]. In 5-year follow-up of our patients, we found that area of anesthesia diminished, but did not completely resolve. Nakajama et al [9] showed that the sural nerve and the lesser saphenous vein have their own independent accompanying arteries. This finding led to the creation of flaps not including the sural nerve, which they called a lesser saphenous veno-adipofascial flap. In our anatomical study and clinical experience, we found that over the course of the sural nerve and the lesser saphenous vein in the posterior leg, the cutaneous artery from the popliteal fossa, the myocutaneous perforators from the sural artery, and the myocutaneous and septocutaneous perforators from the peroneal artery communicate with the accompanying nutrient arteries until they reach the ankle where they communicate with the malleolar vascular network [16]. Both the sural nerve and the lesser saphenous vein have their own accompanying nutrient arteries and vascular supply [19]. Thus, we consider excluding the sural nerve from the flap is feasible. However, the distance between the sural nerve and the lesser saphenous vein varies, and when the distance is small dissection of sural nerve from the flap is difficult and the vascular supply of the flap is prone to injury [19].
We believe the decision to include or exclude the sural nerve should be decided on a case-by-case basis. In cases with a soft tissue defect in the area innervated by the sural nerve (such as defects of the lateral malleolus or dorsal lateral foot), preservation of the sural nerve is not meaningful and a traditional fasciocutaneous flap including the sural nerve should be performed. When separation of the sural nerve and lesser saphenous vein can be performed easily without injury to the vascular supply of the flap, the sural nerve should be excluded from the flap. In cases where the sural nerve is located under the deep fascia until the distal leg, the nerve should not be included in the flap. When the flap is designed beyond the sural nerve territory, the nerve should also be excluded. In cases where the skin defect is in the region innervated by sural nerve, preservation of the ipsilateral sural nerve is insignificant. In rare cases, when the sural nerve is intermingled with its nutrient vessels and separation is impractical, the nerve should not be preserved in order to assure blood supply to the flap.

Theoretically, arterial perfusion and venous reflow positively correlate with flap size and pedicle length, but negatively with pedicle width. Recent reports have indicated success with large peroneal arterial perforator flaps with a pivot point 1 to 5 centimeters above the lateral ankle tip [20, 21]. In our study, we found the distance from the final perforator of the peroneal artery was an average of 2.2 centimeters above the lateral ankle tip. Based on this, we recommended rotating the flap higher than the point 3.5 centimeters above the lateral ankle tip for safety. When the pivot point is moved distally for the repair of defects in the distal foot, more perforators are cut and the vascular supply to the flap is reduced, thus threatening flap survival. In this situation, preoperative Doppler ultrasound examination is advocated to explore the site and determine the diameter and blood flow of the perforator. An intra-operative arterial clamp can also be used to block the perforator to help determine the rotation point. When the sural nerve is excluded from the flap, the pivot point should be located more than 5 centimeters above the tip of the lateral malleolus so that the length of the sural nerve liberated will be shorter and more perforator branches will be preserved. Otherwise, if a longer length of sural nerve is dissected more perforators will be cut threatening flap perfusion. Whenever the sural nerve is excluded it must be carefully detached close to the epineurium, preferably under microscopic guidance. Ligation of lesser saphenous vein must also be performed cautiously to prevent harming the axial vessels of the vein.

The vascular supply system of the distally-based sural flap had been a subject of a great deal of research. It is known that the sural flap is nourished by an uncharacteristic arterial system, and its venous reflow system is not physiological. Arterial perfusion and venous flow of the flap are closely balanced, and the balance is easily affected by postoperative inflammation, compression, and vasospasm which can result in complications such as venous congestion and partial tissue necrosis. Ligation and anastomosis of the lesser saphenous vein can prevent venous congestion of the flap. Postoperative administration of glucocorticoids, dextran-40, and anisodamine can also help to reduce flap congestion, prevent disturbance of venous return, and improve flap circulation.

The primary limitation of this study is the small number of patients. Because of the small number certain statistical comparisons could not be performed. For example, flap area and location of the wound were significantly different between the two groups, and should be controlled for when analyzing post-surgery skin sensation, but this was not possible because of the small sample size. Likewise, the association of postoperative skin sensation with that at 6 months after surgery could not be analyzed because of the small number of patients.

In summary, preservation of the sural nerve is feasible during the creation of a distally-based sural artery flap for foot and ankle reconstruction, and can eliminate loss of sensation of the lateral foot that occur with traditional surgery. Specific criteria should be used to select cases for sural nerve preservation to reduce the risk of flap failure.

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Disclosure of conflict of interest

None.

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